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MASAZUMI HARADA, TADASHI FUJINO, TAKETOSHI AKAGI AND SUSUMU NISHIGAKI

Department of Neuropsychiatry, Institute of Constitutional Medicine, Kumamoto University, Kumamoto,
Department of Neuropsychiatry, Kumamoto University Medical School, Kumamoto,
Department of Physiology, Kumamoto University Medical School, Kumamoto and
Department of Environmental Medicine,
Tokyo Metropolitan Research Laboratory of Public Health, Tokyo

(Received July 28, 1976)

Reprinted from
THE KUMAMOTO MEDICAL JOURNAL
KUMAMOTO JAPAN Vol. 30, No. 2, 1977

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Kumamoto Med. J., Vol. 30, No. 2, 1977 pp. 57–64

MERCURY CONTAMINATION IN HUMAN HAIR AT INDIAN RESERVES IN CANADA

Masazumi Harada*, Tadashi Fujino**, Taketoshi Akagi***
and Susumu Nishigaki****

*Department of Neuropsychiatry, Institute of Constitutional Medicine, Kumamoto University, Kumamoto**,
*Department of Neuropsychiatry, Kumamoto University Medical School, Kumamoto***,
*Department of Physiology, Kumamoto University Medical School, Kumamoto**** and
*Department of Environmental Medicine, Tokyo Metropolitan Research Laboratory of Public Health, Tokyo*****

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Summary. In March and August 1975, a health survey of 89 inhabitants, of whom 71 were examined on mercury content in the hair, was conducted in the province of Ontario, Canada. The highest mercury content in the hair was 80.3 ppm and, among these 71 subjects, 44 showed more than 20 ppm and 23 more than 30 ppm. Mercury values in the hair were positively correlated with the quantity of fish in the diet, and higher in summer when fishes were eaten in larger quantities. These facts lead to the conclusion that mercury contamination of the body is caused by ingesting fishes caught locally.

The authors' limited survey did not reveal typical cases of Minamata disease among the Canadian inhabitants. However, there were 15 cases of glove and stocking type sensory disturbance, and 9 cases of paraconcentric constriction of the visual field. The cases with neurological symptoms showed higher mercury values in the hair than those without the symptoms, and were suspected of mild methylmercury poisoning.

Introduction

In 1970, in the district surrounding the water system of Wabigoon and English rivers in Ontario, Canada, fishes highly contaminated with mercury were discovered (1) (Fig. 1). Later a high content of mercury was also found in fishes and birds of this area and in the mud at the bottom of Clay Lake (2–8). The source of pollution was a chemical plant at Dryden, situated upstream (6). The cat ingesting fishes caught in this water system contracted methylmercury poisoning (9), which could be induced experimentally by feeding the animal with the mercury-contaminated fish (4, 10). In Minamata and Niigata, methylmercury poisoning in cats was observed concurrently with Minamata disease among inhabitants therein (11–15).

Since, in 1970, the above-mentioned circumstances indicated a very dangerous situation, the Provincial Government of Canada prohibited commercial fishing in the area, and examined mercury in the blood as well as in the hair of the inhabitants (9, 16). Actually to determine the mercury pollution, the authors visited the locality in March and August 1975 and conducted epidemiological and clinical surveys as well as analysis of mercury content in the hair. The present communication describes relationships

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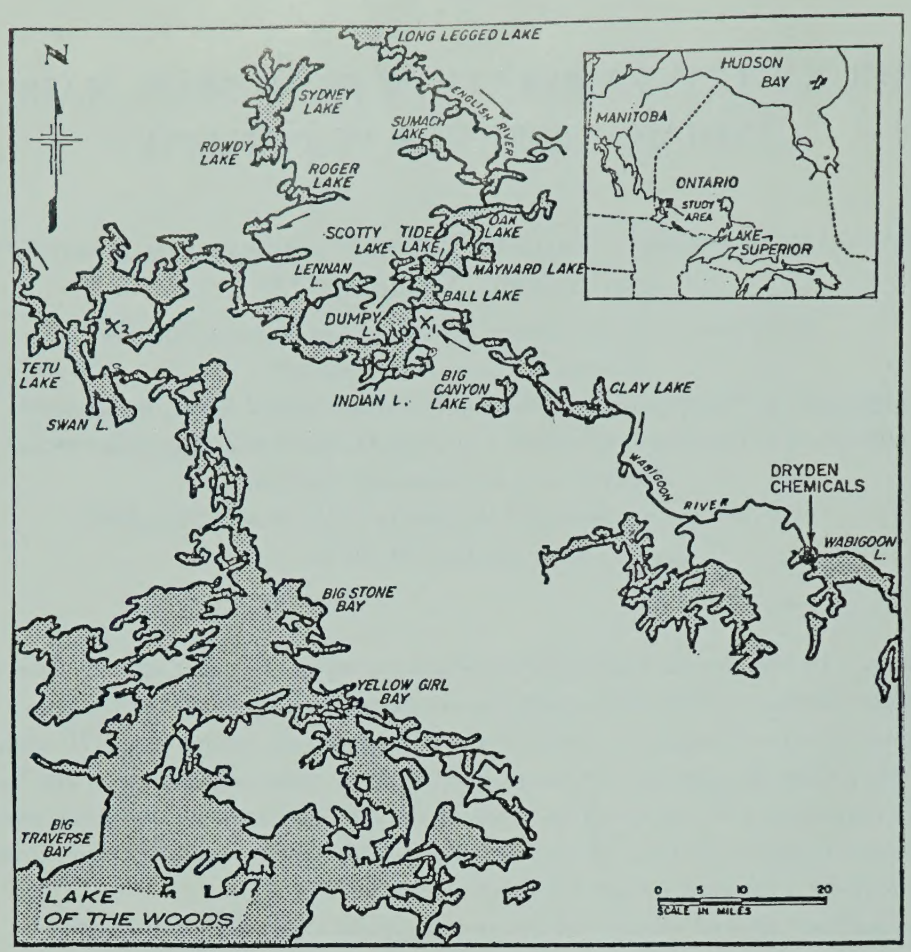

Fig. 1. Sketch map of Wabigoon river system

From Fimreite, N. and Reynolds, L. M.: *J. Wildlife Management*, **37** (1973), 63

X_1: Grassy Narrows, X_2: White Dog.

between mercury contents in the hair and neurological symptoms among the inhabitants.

Methods and Subjects

The health survey on 89 inhabitants of two reserves was conducted by the same methods as applied to the mass examination on Minamata disease in Japan (17,18). The clinical study consisted of routine neurological examination and examination of the visual fields using Förster's perimeter. The subjects for survey were fishing guides, fishermen and their family members. Hair samples of 71 inhabitants were also subjected to measurement of mercury content. The hair sumple was cut as close to the scalp as possible. The sample was cut at 3-cm intervals from the root (the scalp), and mercury content in each section determined by the Cold Vapor Method of atomic absorption spectrophotometry (19); estimating growth of the hair to be 1-cm a month, the degree of mercury pollution can be calculated roughly over three-month period.

Results

Some inhabitants in the regions still showed high mercury values in the hair, in-

dicating a persistent contamination with mercury; for instance, the most proximal segment (the first 3 cm from the scalp) of a hair sample representing the most recent contamination showed a highest value 80.3 ppm. Out of 71 inhabitants, 44 showed more than 20 ppm in the hair and 23 more than 30 ppm.

As shown in Figs. 2–A and 2–B, the mercury level of the hair indicated considerable variations from one subject to another and, moreover, marked fluctuations, even in an individual, from one segment to another. A comparison of mercury levels in the hair between the groups, the one with a dietary habit of continuously ingesting fish (caught in the polluted water system) and the other without such a dietary habit, disclosed a remarkable contrast (Fig. 2–A, 2–B) indicating that the difference in mercury levels was associated with consumption of fishes caught in the polluted river system. Furthermore, the segmental analysis of hair pointed out that the segments corresponding to growth of the hair from March through August had much higher mercury levels than those from September through February. The seasonal fluctuations appeared to

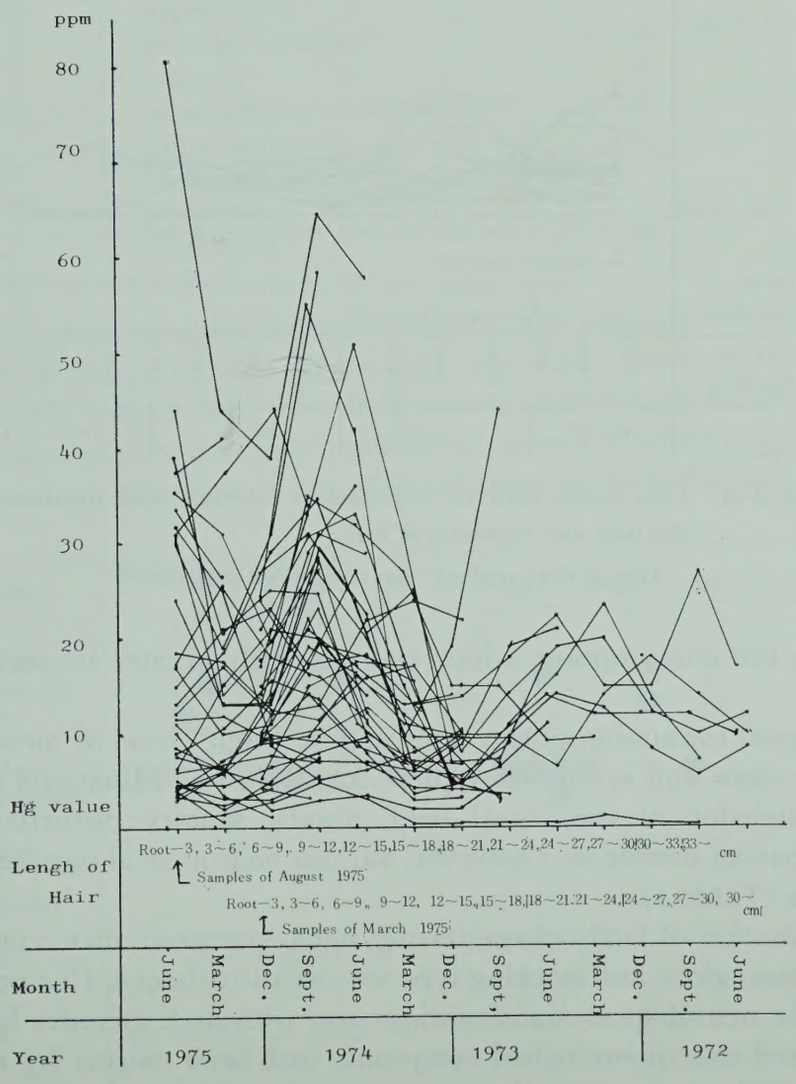

Fig. 2–A. Correlation between intermittent mercury concentration in the hair and ingestion of fish

Group that replied "do eat fish during summer"

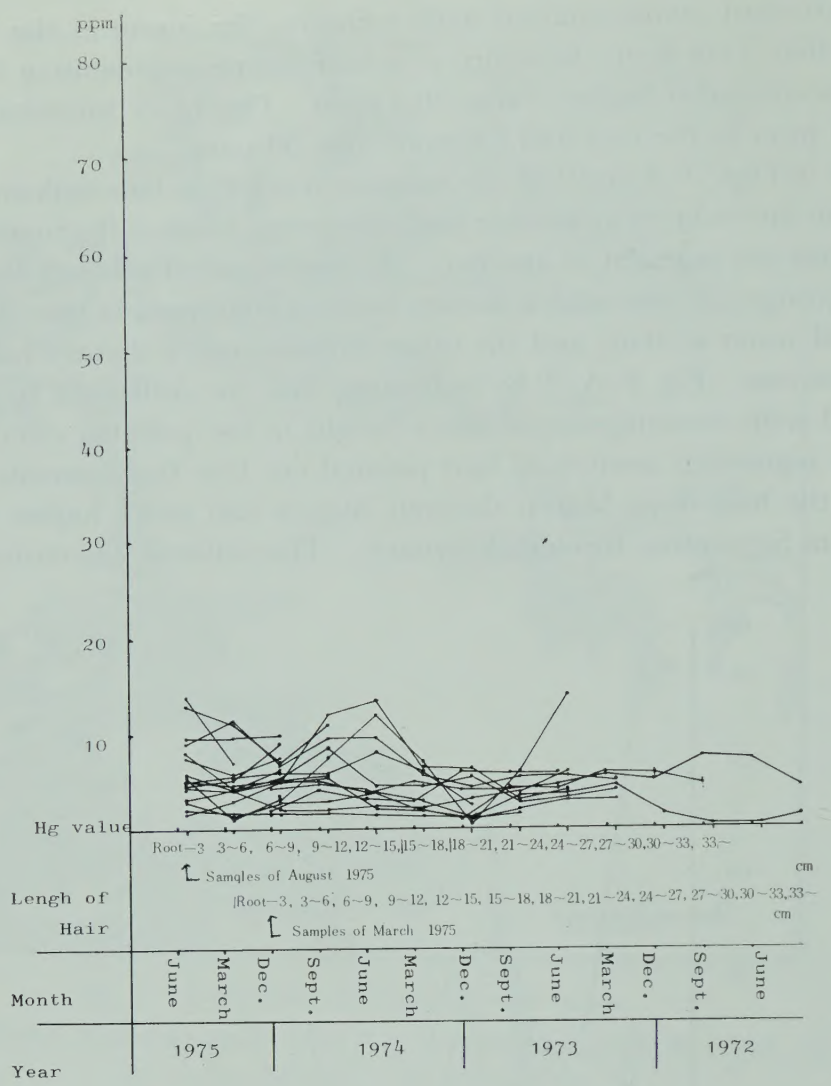

Fig. 2–B. Correlation between intermittent mercury concentration in the hair and ingestion of fish

Group that replied "not eating fish since 1970"

be parallel with fish consumption, which was obviously greater in amount during the hot season.

A neurological evaluation of the cases with a "high" level of mercury in the hair did not disclose signs and symptoms typical (complete) of Minamata disease, such as concentric constriction of the visual field, ataxia, sensory disturbance, disarthria, auditory disturbance, tremor *etc.*; however, various neurological symptoms were found in the habitants (Table 1).

The combination of fairly characteristic signs (paraconcentric constriction of the visual field, 9 cases; glove and stocking type sensory disturbance, 15 cases) with familial occurrence of the neurological manifestations and with high mercury levels in the hair strongly suggested that neurological symptoms had been caused by methylmercury. Some relationship between clinical symptoms and mercury contents in the hair may also be found, as shown in Fig. 3.

In the following groups with objective symptoms (A, B, C), mercury values in

TABLE 1. *Clinical symptoms (89 cases)*

		Number of cases
Subjective symptoms (complaints)	Numbness	28
	Pain in limbs	40
	Convulsion of limbs (Cramps)	16
Neurological symptoms	Disturbance of eye movement	19
	Nystagmus	3
	Impaired hearing	40
	Sensory disturbance	37
	(glove and stocking type)	(15)
	(perioral area)	(5)
	Abnormalities in the visual field	16
	(para-concentric contraction)	(9)
	Tremor	21
	Hyporeflexia	20
	Hyperreflexia	15
	Muscular weakness	16
	Ataxia	8
	(when walking on straight line)	(8)
	(adiadokokinesis)	(16)
	(dysmetry)	(7)
	Dysarthria	5
	Fainting-fit	2

the hair were higher than those for inhabitants with only subjective symptoms (D) or none at all (E); these groups include A) patients with abnormality of the visual fields (inclusive of concentric constriction) as well as sensory disturbance, B) those with other neurological symptoms such as auditory impairment as well as sensory disturbance, and C) those without sensory disturbance but with neurological disturbance (Fig. 3).

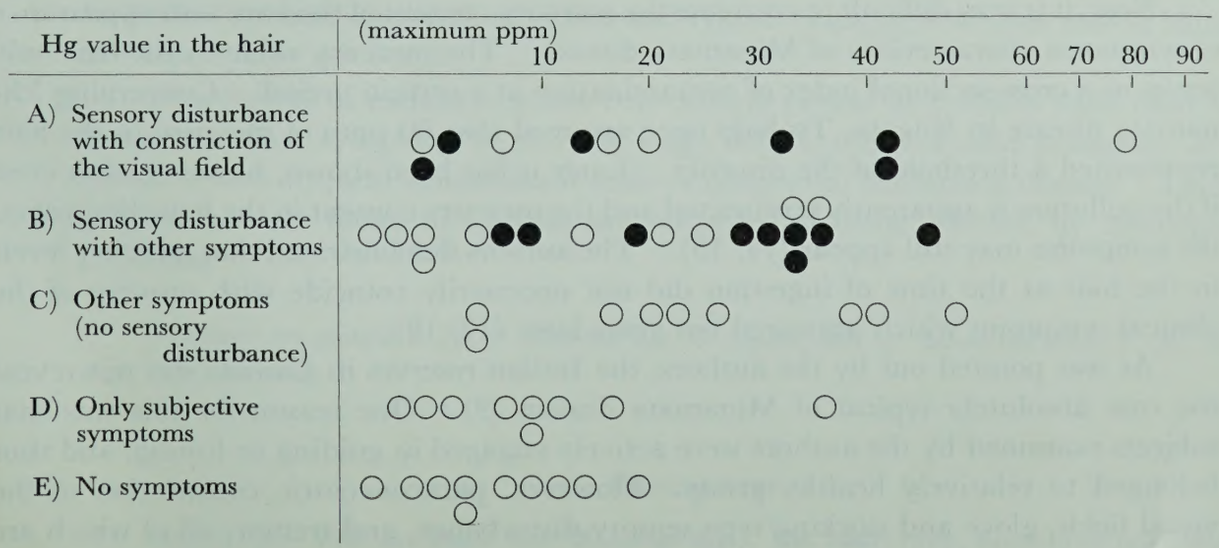

Fig. 3. Correlation between clinical symptoms and mercury value in the hair

● : Sensory disturbance of glove and stocking type

○ : Sensory disturbance of other types

Among the cases with glove and stocking type sensory disturbance (solid circles in Fig. 3), mercury content in the hair was more than 20 ppm in 10 out of 15 cases.

Discussion

Analysis of mercury in the hair has shown that inhabitants of the two Indian reserves are still being contaminated with mercury. The mercury content in the hair was high in the inhabitants who ate relatively large quantities of fishes, or who were engaged in fishing or guiding for sport fishing. The mercury value is higher in summer when fishes are more ingested.

Detection of mercury from the hair, blood or urine is often effective in diagnosing mercury poisoning. An important clue to the cause of Minamata disease in Japan was the discovery of high mercury values in the samples from patients. As was the case of Minamata disease in Japan, the contamination with mercury of the inhabitants at the reserves was continuous over a long period of time (12–15, 18). In both instances of environmental pollution in Japan and Canada, mercury values in the hair which change slowly provide a more reliable clue than such values in the urine or blood changing rapidly. Accordingly, successive analyses of mercury in the hair were effective in determining the actual degree of contamination among inhabitants, as demonstrated in this study.

Clarkson who simultaneously presented similar results on the reserves to those of the authors showed seasonal changes in, and higher values of mercury content of the hair of the inhabitants (maximum 105 ppm), for instance, a fishing guide and his family with a high mercury content in the hair and with similar seasonal changes in mercury values among family members. This led him to the belief that the mercury contamination was due to food intake (20). Clarkson also compared the mercury content of the hair with that of the blood: 30 ppm in the hair and, in contrast, about 100 ppb in the blood (20).

Now, it is very difficult to correlate the mercury content of the hair with appearance of symptoms characteristic of Minamata disease. The mercury value in the hair only serves as a cross-sectional index of contamination at a certain period. Concerning Minamata disease in Niigata, Tsubaki once assumed that 50 ppm of mercury in the hair represented a threshold of the disorder. Later it has been shown, however, that even if the pollution is apparently terminated and the mercury content in the hair diminishes, the symptoms may still appear (14, 15). The authors demonstrated that mercury levels in the hair at the time of ingestion did not necessarily coincide with severity of the clinical symptoms which appeared ten years later (17, 18).

As was pointed out by the authors, the Indian reserves in Canada did not reveal the case absolutely typical of Minamata disease (9). One reason for this was that subjects examined by the authors were actively engaged in guiding or fishing, and thus belonged to relatively healthy group. However, paraconcentric constriction of the visual fields, glove and stocking type sensory disturbance, and tremor, all of which are seen in patients of Minamata disease, were also detected at the reserves (9). Though the mercury content in the hair is of limited clinical value, its levels were higher in the cases with the symptoms than in those without them (14, 15, 18).

The mercury contamination in Canada is less in its extent than that in Minamata, and some certain differences exist in manifestations of the contamination between the two localities. In Minamata 100,000 people live in an area in size nearly equal to the two reserves in Canada. Inhabitants of Minamata ingest fish and shellfish throughout the year, and they do not have such a strong liking for alcohol (21). In Minamata the source of pollution exists in densely populated area, and many people have lived around the outlet of effluent. It is said that mercury, in amount of 600 tons, was discharged from the outlet (18). The plant was producing acetaldehyde and methylmercury discharged directly into the sea (12, 13, 18). In case of Canada, the population is sparse, the source of pollution is far from the reserves, and in winter the lakes and rivers freeze so that the intake of fish is greatly reduced. The factory produces caustic soda on a relatively smaller scale, 10 tons of mercury were discharged from there, and inorganic mercury was being discharged (2, 6).

The mechanism of methylation of inorganic mercury still remains unanswered (22), but more than 80% of mercury detected in fishes from the Wabigoon river was methylmercury (5). The fish obtained at Clay Lake was fed to cats, which experimentally induced methylmercury poisoning (5, 10).

In short, differences in the ratio of morbidity and severity of symptoms are found between the cases in Canada and Japan, but there is no essential dissimilarity in them.

It was the report of Hunter and Russell which first suggested that Minamata disease studied at that time coincided, clinically and pathologically, with those reported in Hunter's paper (23, 24). However, as the extent of polluted areas was fully exposed, it was realized that such typical and serious cases were likely to be only tips above sea level of icebergs (18).

It has become clear that atypical (incomplete) and mild cases vastly outnumber typical and severe ones. In some patients, symptoms were so mild that only sensory disturbance was observed, where diagnosis could not have been decisive unless typical cases were found among their family members, or mercury contamination was proven. In the future, mass outbreaks of serious and typical cases, such as those seen in Minamata or Niigata, may not occur.

If pollution of mercury occurs over a long period at a lower level than that in Minamata Bay, symptoms at the first stage will be those of mild poisoning, as in incomplete and atypical Minamata disease or congenital Minamata disease. Thus, such patients as were found in Canada are suspected of being affected by methymercury poisoning.

The authors are grateful to Dr. P. Newbery, Mr. D. Barnes, Mrs. C. Barnes and Miss T. Jill for useful assistance to this survey.

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